

## A Case of Primary Bacterial Pericarditis with Recurrent Cardiac Tamponade

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Cardiac tamponade is an important and potentially lethal complication of acute pericarditis. However, recurrence of cardiac tamponade is rare when it is treated appropriately. We present a 49-year-old man with bacterial pericarditis and recurrent cardiac tamponade, which was caused by the rupture of an upper part of the left atrium (LA). According to the autopsy findings, bacteremia from *Staphylococcus aureus* developed on a substrate of poorly controlled diabetes mellitus and spread to the pericardium via the blood. Subsequently, tissue necrosis developed from the pulmonary trunk and aorta to the LA, leading to recurrence of cardiac rupture and cardiac tamponade. (J Nippon Med Sch 2017; 84: 133–138)

**Key words:** bacterial pericarditis, recurrent cardiac tamponade

### Background

Cardiac tamponade is an important and potentially lethal complication of acute pericarditis. However, recurrence of cardiac tamponade is rare when it is treated appropriately<sup>1</sup>. In the present report, we describe an extremely rare case of bacterial pericarditis with recurrent cardiac tamponade, which was caused by the rupture of an upper part of the left atrium (LA). This uncommon pathological condition and its clinical course are discussed in relation to the autopsy findings.

### Case Presentation

A 49-year-old man presented to our emergency department with a two-week history of sore throat, sneezing, and worsening dyspnea. He had diabetes and hypertension but he had taken no medication, including insulin injections, for about one year.

At admission, blood pressure was 86/67 mmHg, heart rate was 121/min, respiratory rate was 48/min, oxygen saturation was 100% with 6 L/min oxygen, and body temperature was 38.9°C. His level of consciousness was clear. No heart murmurs, lung crackles, or pericardial rub sounds were heard on auscultation, but his heart sounds were diminished. No leg edema was visible, but the patient's jugular vein was distended. His blood results demonstrated severe inflammation (white blood

cells, 18,200/ $\mu$ L; C-reactive protein, 30.1 mg/dL; procalcitonin, 2.57 ng/mL), renal dysfunction (blood urea nitrogen, 31.3 mg/dL; creatinine, 1.3 mg/dL), liver dysfunction (aspartate aminotransferase, 370 IU/L; alanine aminotransferase, 311 IU/L; total bilirubin, 1.6 mg/dL), mild anemia (Hb, 12.5 g/dL), mild abnormality of coagulation/fibrinolysis system (platelets,  $340 \times 10^3$  / $\mu$ L; prothrombin time international normalized ratio, 1.76; activated partial thromboplastin time, 36.9 s; D-dimer, 3.7  $\mu$ g/mL), and uncontrolled diabetes (blood sugar, 807 mg/dL; hemoglobin A1c, 11.8%). Creatine kinase, creatine kinase-MB and cardiac troponin-T were within their normal ranges. Brain natriuretic peptide level was slightly elevated (brain natriuretic peptide, 69.4 pg/mL). The blood gas examination revealed metabolic acidosis (pH 7.33, B.E. -11.3) and hyperlactatemia (lactate 73 mg/dL). The chest radiograph showed cardiomegaly (cardiothoracic ratio, 71%) but no congestion (**Fig. 1a**). The electrocardiogram showed sinus tachycardia, at 117 beats per minute, and ST elevation in leads I, II, III, aVF, and V2–6 (**Fig. 1b**). Transthoracic echocardiography showed a massive pericardial effusion (13–23 mm) and a dilated inferior vena cava (20 mm, no respiratory change), which indicated cardiac tamponade. Left ventricular ejection fraction was normal and no valve regurgitations were seen (**Fig. 2a**). Computed tomography

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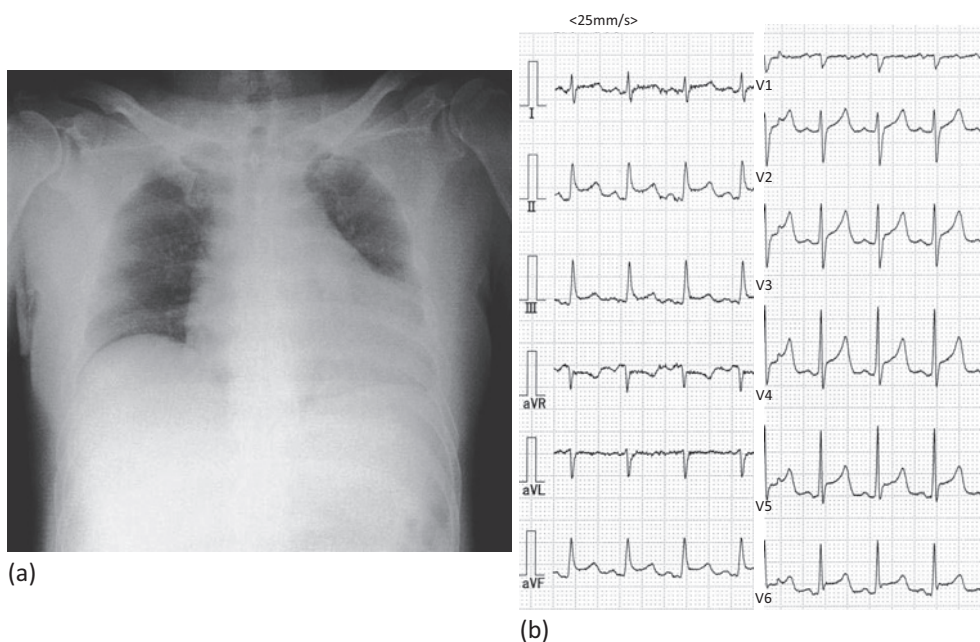


Fig. 1 (a): The chest radiograph showed cardiomegaly (cardiothoracic ratio, 71%) but no congestion.  
 (b): Electrocardiogram (ECG) obtained on admission. The ECG during the sinus tachycardia showing ST elevation in leads I, II, III, aVF, and V2–6.

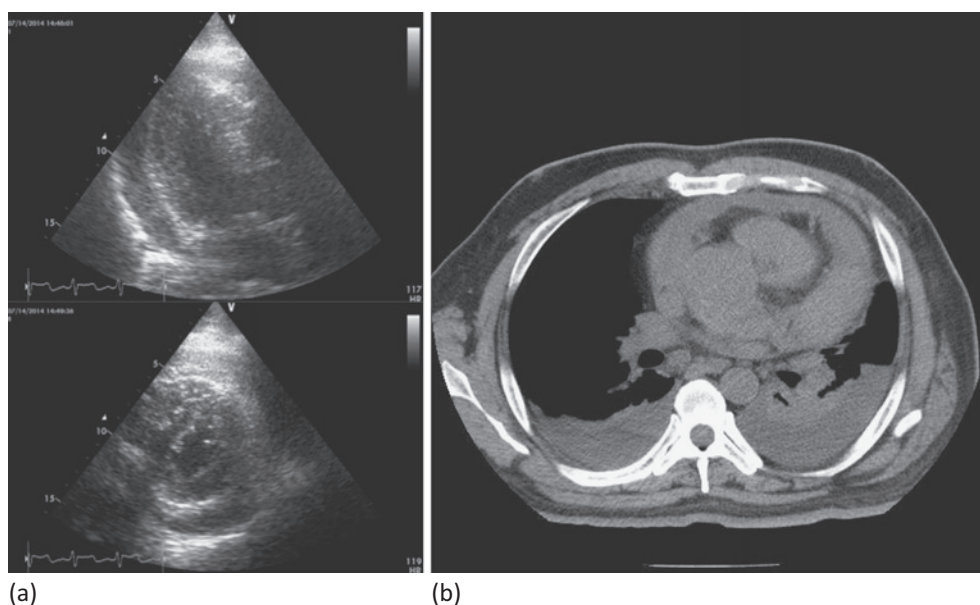


Fig. 2 (a): Transthoracic echocardiography showed a massive pericardial effusion (13–23 mm) and a dilated inferior vena cava (20 mm, no respiratory change). Left ventricular ejection fraction was normal and no valve regurgitations were seen.  
 (b): Chest computed tomography (CT) scan taken on admission showing massive pericardial effusion and bilateral pleural effusions. The CT attenuation value of the effusion was high (30 IU).

(CT) without contrast media was performed to investigate the focus of inflammation, but no marked abnormalities were seen, apart from the pericardial effusion.

The CT attenuation value of that effusion was high (30 IU), which indicated blood (Fig. 2b).

In view of the obstructive shock due to cardiac tam-

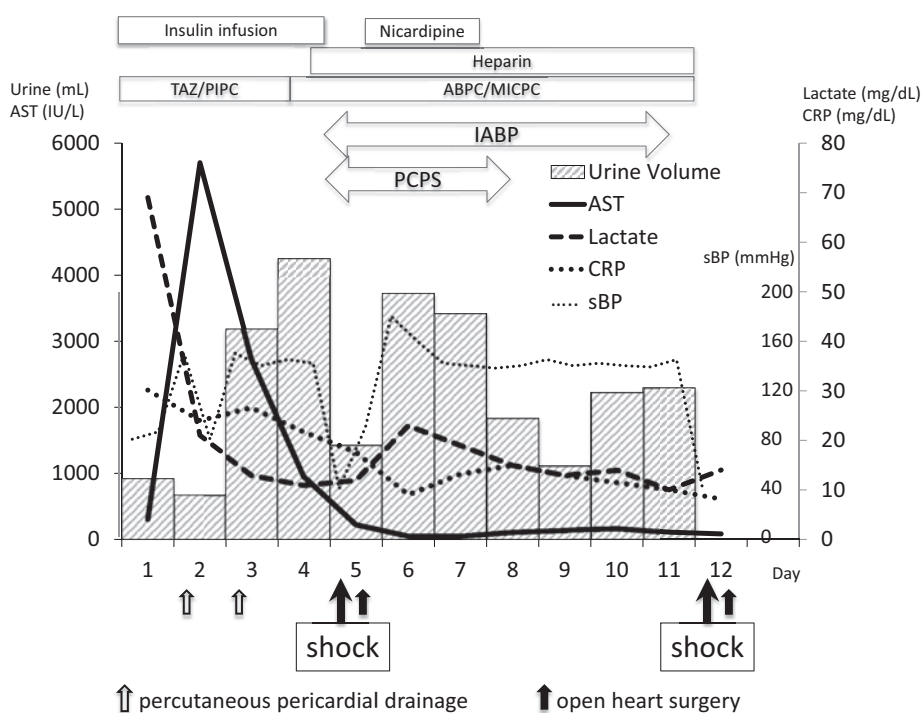


Fig. 3 Clinical course of urine volume, laboratory (aspartate aminotransferase, lactate, C-reactive protein), systolic blood pressure (sBP), and therapies.

ponade, diagnostic and therapeutic pericardiocentesis was performed, after which the patient’s hypotension and tachycardia were normalized. At the same time, we started continuous infusion of insulin for hyperglycemia and empirical antibiotic therapy with tazobactam/piperacillin for suspected severe infection. On the second day, *Staphylococcus aureus* was identified from both blood and pericardial effusion cultures. Accordingly, we diagnosed bacteremia and bacterial pericarditis and we changed the antibiotic regimen from tazobactam/piperacillin to ampicillin/cloxacillin, which is very effective for *Staphylococcus aureus*. On the fifth day, there was a sudden deterioration in the patient’s clinical status, due to recurrence of the cardiac tamponade despite the continuous drainage, so we started percutaneous cardiopulmonary support (PCPS) and inserted an intra-aortic balloon pump and transferred him to the operating room. During the heart opening, a bleed from the upper part of the LA was identified and was sutured. The patient was taken off PCPS on the eighth day and was also taken off intra-aortic balloon pump therapy on the tenth day. His inflammatory state gradually improved. However, there was a sudden cardiac deterioration, again due to cardiac tamponade recurrence, on the eleventh day. We tried reintroducing PCPS and opened the heart again to stop the bleeding but it was difficult and the patient died. His

clinical course is shown in **Figure 2**. At autopsy, hematomas from the left side of the aorta to the upper part of the LA, which contained gram-positive cocci, and necrotic tissue in the LA, were found (**Fig. 3, 4**). An infarct lesion was seen in the LA appendage, but it did not contain any bacterial infection. No signs of endocarditis were seen and no other abnormal findings were seen in other organs.

Verbal informed consent was obtained from the patient’s parent for publication of this case report.

### Discussion

We describe an extremely rare case of bacterial pericarditis with recurrent cardiac tamponade, which was caused by the rupture of part of the upper LA. According to the autopsy findings, bacteremia from *Staphylococcus aureus* developed on a substrate of poorly controlled diabetes mellitus and spread to the pericardium via the blood. The cause of the bacteremia was not clear, because no foci of infection were found on autopsy. Subsequently, tissue necrosis developed from the pulmonary trunk and aorta to the upper part of the LA, leading to cardiac rupture and cardiac tamponade.

Cardiac tamponade recurrence, in this case caused by the LA rupture due to bacterial pericarditis, is very rare and difficult to diagnose<sup>2</sup>. There are some previous re-



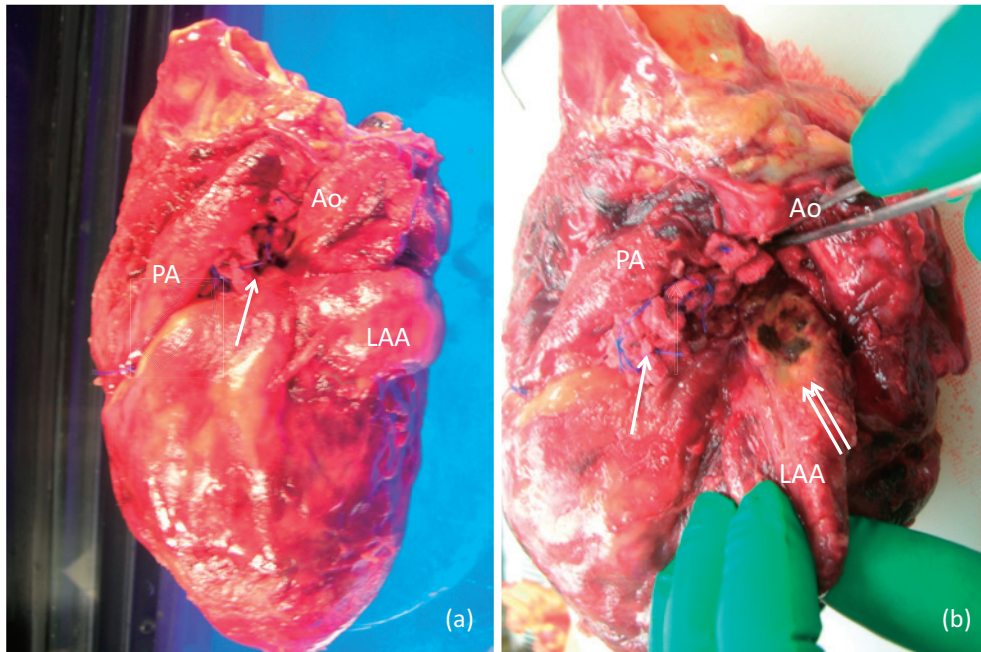


Fig. 4 The autopsied heart.

(a): The ruptured and sutured site is indicated by the arrow.

(b): Top view, showing the sutured site from the pulmonary artery trunk to the basal aorta (**arrow**) and the hemorrhagic and necrotic tissue of an upper site in the left atrium (**double arrows**).

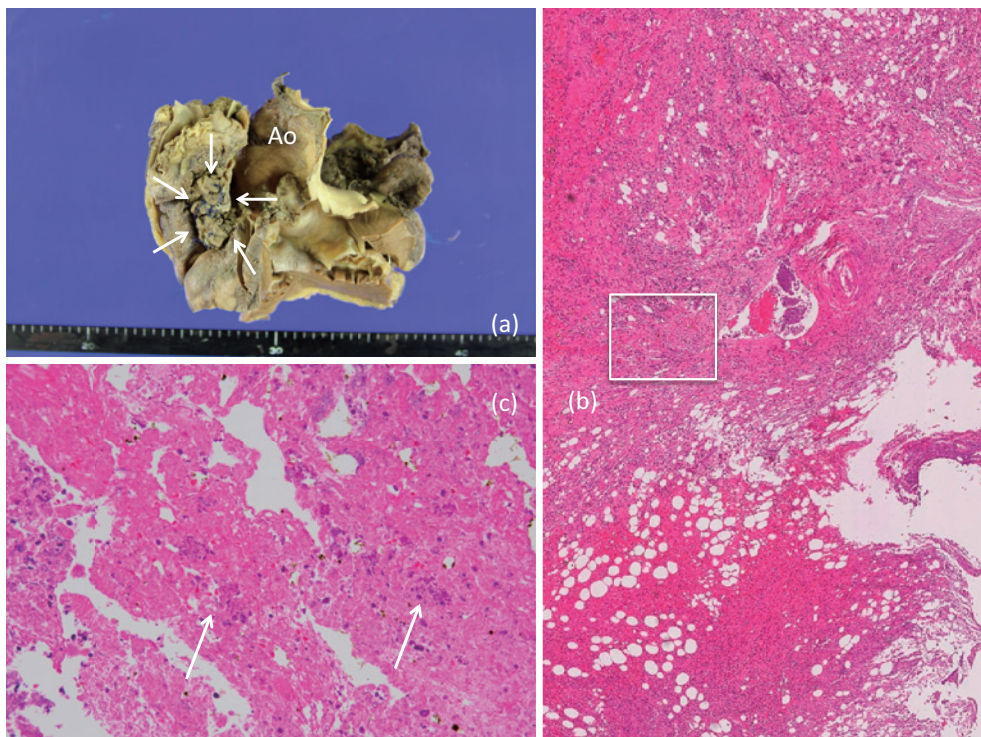


Fig. 5 (a): Hematoma from the left side of the aorta to an upper area of the left atrium (**arrows**).

(b): Microscopic findings of hematoma in (a). Coagulation with neutrophil accumulation and foci of bacterial colonies are seen.

(c): Focused view of square in (b). Bacterial colonies are indicated by arrows.

Table 1 Cases of Primary Bacterial or Purulent Pericarditis with Cardiac Rupture

No.	Author	Published	Cause of pericarditis	Pathogens	Part of rupture	Outcome	Reference
1	Juneja R	1999	Bacteremia	<i>S.aureus</i>	LV	alive	5
2	Osula S	2001	Bacteremia	<i>S.aureus</i>	LV	alive	6
3	Chen AK	2010	Bacteremia	<i>S.aureus</i>	LV	alive	7
4	Krishna MR	2015	Bacteremia	<i>S.pneumoniae</i>	LV	alive	8
5	Sunkara B	2015	Unknown	<i>S.viridans</i>	LV	dead	9
6	Yoshizane T		Bacteremia	<i>S.aureus</i>	LA	dead	present case

Abbreviations: LV; left ventricular, LA; left atrium.

ports of cardiac rupture due to bacterial pericarditis. Most of those cases were associated with endocarditis, aortic dissection and trauma<sup>3,4</sup>. Endocarditis and aortic dissection were not seen at autopsy in our patient, whereas the LA rupture was seen during the first surgery and the tissue necrosis of the upper part of the LA was seen at autopsy; we thus concluded that LA rupture due to bacterial pericarditis occurred. There were only 6 cases of cardiac rupture due to primary bacterial or purulent pericarditis with cardiac rupture, excluding endocarditis, aortic dissection, infectious aortic aneurysm, ischemic heart disease and trauma<sup>5-9</sup>, (Table 1). Surgery and antimicrobial therapy were performed in all cases. To our knowledge, this is the first case of LA rupture due to bacterial pericarditis to be reported in the English language literature.

There are a number of possible reasons for the recurrence of cardiac tamponade. First, the *Staphylococcus aureus* could have caused the tissue destruction in the subacute phase, although the medical treatment for the infection might have been responsible. The usual pathogens in bacterial pericarditis are *Staphylococcus aureus* (36%), *Streptococcus pneumoniae* (21%), and *Haemophilus influenzae* (12%), but mortality is highest among those with *Staphylococcus aureus* infection<sup>10,11</sup>. In our patient, we believe that the infected tissue was severely damaged because the *Staphylococcus aureus* was very tissue-destructive<sup>6</sup>. Hence, the unexpected rupture occurred again in the subacute phase. Second, bacterial pericarditis occurs frequently in individuals with predisposing conditions, such as previous cardiac surgery, chest trauma, dialysis, alcoholism, and immunosuppression<sup>12</sup>. We assumed that our patient might be in an immunosuppressed condition because he had uncontrolled diabetes mellitus<sup>13</sup>. This might have led to the bacteremia and bacterial pericarditis. Any other abnormal findings for immunosuppression such as endocrine disorder or malnu-

trition were not seen. Finally, we could not identify the location of the cardiac rupture accurately.

### Conclusions

LA rupture due to bacterial pericarditis is very rare and difficult to diagnose. Echocardiography and plain-CT were difficult to evaluate the LA. Enhanced-CT would be necessary for a more accurate diagnosis.

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**Conflict of Interest:** The authors declare that they have no competing interests.

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